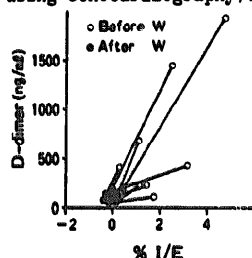


**SIMPLE ACCESS FOR HEMATOLOGICAL ACTIVITY OF INTRACARDIAC THROMBI BY D-DIMER ASSAY**

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To clarify whether plasma level of fibrin specific degradation product, D-dimer, could reflect the hematological activity of intracardiac thrombi, D-dimer and  $\text{XI/E}$  (the ratio of indium-111 radioactivity accumulated on thrombi to that in the blood pool using dual-tracer technique) were measured in 31 patients with intracardiac thrombi (detected using echocardiography). D-dimer values closely correlated with those of  $\text{XI/E}$  ( $r=0.712$ ,  $p<0.01$ ). Eight patients with high level of  $\text{XI/E}$  received 2-4mg/day of warfarin (W) for 6-12 weeks. After administration of warfarin, the plasma level of D-dimer significantly decreased from  $675.6 \pm 231.8$  to  $111.9 \pm 20.5$  ng/ml with a significant decrease in  $\text{XI/E}$  ( $2.03 \pm 0.44$  to  $-0.16 \pm 0.03$ ), as shown in figure. These results strongly suggested that the hematological activity of intracardiac thrombi could be quantitatively assessed by the simple measurement of D-dimer, and that such a measurement can be useful to evaluate the effects of anticoagulant therapy repeatedly in patients with hematologically active thrombi.

**CYCLE LENGTH VARIABILITY PREDICTS CARDIAC MORTALITY INDEPENDENT OF DILTIAZEM THERAPY**

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In MDPIT, Holter recordings were obtained during the hospitalization for the index acute myocardial infarction (AMI). We were able to estimate the cycle length variability (CLV, defined as the standard deviation of the R-R intervals over the 24-hour recording) for 1576 of the 2466 patients randomized to diltiazem or placebo.  $\text{CLV} \leq 50$  msec was observed in 142 (9.0%),  $50 < \text{CLV} \leq 100$  msec in 1004 (63.7%) and  $100 < \text{CLV} \leq 200$  msec in 430 (27.3%). At study termination (4.3 years) the estimated product limit cardiac death rates were 32.1% for those with  $\text{CLV} \leq 50$ , 7.7% for those 50-100, and 6.6% for those 100-200. The increased risk for those with  $\text{CLV} \leq 50$  (Cox hazard ratio=3.88, 2.18-6.89, 95% confidence interval) was the same in both the diltiazem and placebo treated groups. In contrast to the MDPIT prior findings with respect to indices of left ventricular dysfunction there was no bidirectional interaction of diltiazem therapy and low CLV. Even controlling simultaneously for age, antiarrhythmic therapy, beta blocker therapy, non-Q-wave AMI, NYHA functional class, prior MI, pulmonary congestion on X-ray during the AMI, rates in the CCU, diltiazem therapy and the interaction of diltiazem therapy and pulmonary congestion, the partial hazard ratio was 1.85 (98-3.51). We also computed an average heart rate for each hour and computed the standard deviation of these heart rates as an estimate of the ultra-low frequency (e.g. circadian) changes in heart rate. Inclusion of this index into the Cox model did not reduce the added risk for low CLV. We conclude that the risk associated with low CLV is more likely related to higher frequency components such as respiratory sinus arrhythmia and is independent of poor left ventricular function and diltiazem therapy.

Wednesday, March 21, 1990

Poster Displayed: 9:00AM-12:00NOON

Author Present: 11:00AM-12:00NOON

Hall C, New Orleans Convention Center

The Electrocardiogram: Arrhythmias and Prognosis

**ELECTROCARDIOGRAPHIC ABNORMALITIES IN COCAINE USERS: A PROSPECTIVE STUDY**

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Experimental and clinical evidence suggests a link between cocaine use and myocardial ischemia; this association has not been studied in a prospective fashion. Between August 1988 and February 1989, 49 active crack cocaine users were studied at the Detroit Receiving Hospital Emergency Department (ED). 12-lead electrocardiograms (ECG) were performed and compared to ECGs of a matched control group (n=40) of non-users admitted to the same ED. ECGs were reviewed by investigators blinded to group assignment. ST-T wave abnormalities judged to be ischemic were observed in 9 patients (18%) in the cocaine group; 4 had anginal symptoms. Significant QTc (msec) prolongation was observed in 16% of cocaine users. Data are shown as mean  $\pm$  SEM.

	age(years)	males	ST-T wave abnormalities	QTc
Cocaine users	30 $\pm$ 1	29	9	430 $\pm$ 4
Non-users	32 $\pm$ 1	27	0	418 $\pm$ 3
p	N.S.	N.S.	0.004	0.03

We conclude that crack cocaine use is associated with significant QTc prolongation, and with silent as well as symptomatic ischemic ECG abnormalities.

**EFFECT OF DAILY ACTIVITIES ON THE CIRCADIAN RHYTHM OF MYOCARDIAL ISCHEMIA OUT-OF HOSPITAL.**

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It is not known whether the circadian rhythm of myocardial ischemia is "exogenous" (resulting from patient activities) or "endogenous" (independent of patient activities). Thus, we employed a structured diary system in 39 CAD patients with evidence of transient ischemia during 4-48 hours Holter monitoring. There were 118 ischemic (I) episodes with an average duration (dur) of  $8.7 \pm 10$  min. Idur was related to the intensity of physical (Phys;  $p<.05$ ) and mental (Ment;  $p<.09$ ) activities. The number and dur of I episodes showed a marked circadian rhythm with greatest Idur within 6 hr of waking (timeblock 1); similarly, the intensity of Phys and Ment activities (assessed on a 6-point scale) showed a circadian rhythm:

6hr timeblock:	1	2	3	4	5	6
I(min)	13.7 $\pm$ 3	8.2 $\pm$ 12	4.8 $\pm$ 9	0.5 $\pm$ 1	.001	
Phys score	3.2 $\pm$ 3	3.0 $\pm$ 5	2.4 $\pm$ 9	1.7 $\pm$ 1	.001	
Ment score	4.1 $\pm$ 7	4.1 $\pm$ 6	3.1 $\pm$ 8	2.1 $\pm$ 1	.001	

After controlling for patient activity levels using analysis of covariance, there was no longer evidence for a circadian rhythm for ischemia. **CONCLUSION:** The circadian rhythm for myocardial ischemia appears to be an exogenous phenomenon related to physical and mental activity levels in CAD patients.